A DISSERTATION ON

“SMALL BOWEL PERFORATION IN ACUTE ABDOMEN - REDEFINING ITS CAUSE”

Dissertation submitted to

THE TAMIL NADU Dr.M.G.R.MEDICAL UNIVERISTY

CHENNAI

with partial fulfilment of the regulations for the Award of the degree

M.S. (General Surgery)

Branch – I

INSTITUTE OF GENERAL SURGERY,

MADRAS MEDICAL COLLEGE,

CHENNAI.

MAY - 2019

CERTIFICATE

This is to certify that the dissertation entitled **"SMALL BOWEL PERFORATION IN ACUTE ABDOMEN -REDEFINING ITS CAUSE"** is a bonafide work done by **DR.SUJEETH AROUMOUGAME**., in partial fulfilment of the requirements for M.S.Branch– I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in May 2019.

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DECLARATION

I hereby solemnly declare that the dissertation titled **"SMALL BOWEL PERFORATION IN ACUTE ABDOMEN - REDEFINING ITS CAUSE"** is done by Me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai under the guidance and supervision of Prof.Dr.ALLI DGO, M.S. The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfillment of requirements for the award of M.S. Degree (Branch-I) in General Surgery.

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Dear Dr.A.Sujeeth,

The Institutional Ethics Committee has considered your request and approved your study titled **"SMALL BOWEL PERFORATION IN ACUTE ABDOMEN – REDEFINING ITS CAUSE "** - **NO.10062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD.,	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	:Member Secretary
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10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

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LIST OF ABBREVIATIONS USED

AIDS	Acquired Immunodeficiency Syndrome
AUD	Air under diaphragm
AFB	Acid fast bacilli
Bx	Biopsy
C/S	culture/Sensitivity
CSF	cerebrospinal fluid
dL	decilitre
DNA	deoxyribonucleic acid
DOA	date of admission
DOD	date of discharge
DOS	date of surgery
E. coli	Escherichia coli
Ef	Enteric fever
gm	grams
GB	gall bladder
GI	gastrointestinal
Hb	haemoglobin
Hg	mercury
Kleb	klebseilla
L	litres
mg	milligrams
NT	Not taken
NSP	No specific pathology
Perf	Perforation
Salm	Salmonella
TB	Tuberculosis

ABSTRACT

Background & Objectives:

Small bowel perforation is a common problem seen in acute abdomen. The commonest cause being typhoid fever followed by tuberculosis and other cause.

Aims & Objectives:

To analyse and compare the cause of small bowel perforation in acute abdomen

Methods:

The study was conducted in Institute of General Surgery, Madras Medical College, Chennai from MAY 2017 TO OCTOBER 2018. A minimum of 103 patients of Small bowel perforations included in the study. Patients with traumatic perforations, Large bowel perforation duodenal perforation (D1 AND D2) and irradiated abdomen have been excluded. Factors were tabulated and statistically analysed to study their contributions.

Results:

The study concludes the commonest cause of Small bowel perforation was typhoid followed by non specific causes. Perforation commonly occurred in the third and fourth decade of life with patients between the ages of 30 and 50. Pneumoperitoneum in chest x-ray and erect abdominal x-ray was seen in 80% of patients.

Conclusion: Typhoid is the most common cause of small perforation, followed by non-specific perforation. Other Causes of small bowel perforation include non-specific, TB, Widal test is helpfull in the diagnosis of typhoid fever.

TABLE OF CONTENTS

INTRODUCTION

INTRODUCTION

Enteric fever is endemic in developing countries, including India. The incidence of intestinal perforation in cases of typhoid fever is about 2-3%. Widal test, although done routinely, has been found to be non-specfic and difficult to interpret in areas where typhoid fever is endemic. Diagnosis of typhoid is rarely confirmed and in the majority of cases of enteric perforation, only a conjectural diagnosis is based on the circumstantial evidence of terminal ileal, antimesenteric perforation in an adult running fever for two weeks. In India, typhoid is seen in 8-50% cases of gastrointestinal perforation.

The proximal intestinal perforations are more common in India as compared to the distal intestinal perforations, which are more frequent in developed countries. Site and etiological factors of perforations also show geographical Variations. Therefore, there is a need to study this problem, especially in a typhoid-endemic country like India, where the burden of ileal perforations is high and there is convincing evidence that surgical intervention and definitive antibiotic therapy can decrease mortality.

This study was undertaken to determine the etiological diagnosis, appropriate therapeutic approach of ileal perforation and to re-visit the concept of .enteric perforation..

AIMS AND OBJECTIVES

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- The aims and objectives of this study are
- To analyse and compare the cause of small bowel perforation in acute abdomen

REVIEW OF LITERATURE

REVIEW OF LITERATURE

History

Small bowel perforation is a serious complication of a variety of Diseases. Typhoid fever was the most common cause.

History of surgery is as old as history of mankind. Hippocratic facies which represents terminal stage of peritonitis have been recognized since Hypocrites 400BC. The first published report of perforated duodenal ulcer was by Hanbergery in 1746.

Hippocrates coined the term typhus (gr. cloudy) in 460 B.C [(6)]. In 1829 Louis later term typhoidae and discussed 150 cases with intestinal perforation, haemorrhage, spleenomegaly, rose spots and mesenteric lymphadenopathy [(7)].

Karl Joseph Ebereth discovered the Typhoid bacillus in 1880. In 1884, Gaffkey first isolated and cultured *Salmonella typhi* [(7)].Widal described the test to detect agglutinins in the serum of patients suffering from typhoid fever in 1896. The first vaccine for human use against typhoid was made by Pfeiffer and Kalle in 1896 [(7)].

Surgery for the treatment of typhoid perforation began in late 1800's. After work of Finney and Cushing, surgery became the standard treatment. Typhoid was also known in ancient Indian medicine – Ayurveda as Sannipath Jawara and perforation as Chidrodra. This detailed description was present in Sushrutha Samhita.

ANATOMY

Small intestine extends from pylorous to ileocaecal junction. It is about 6m long.

It is divided into

1. Upper fixed part called – Duodenum
2. Lower mobile part – Jejunum and Ileum.

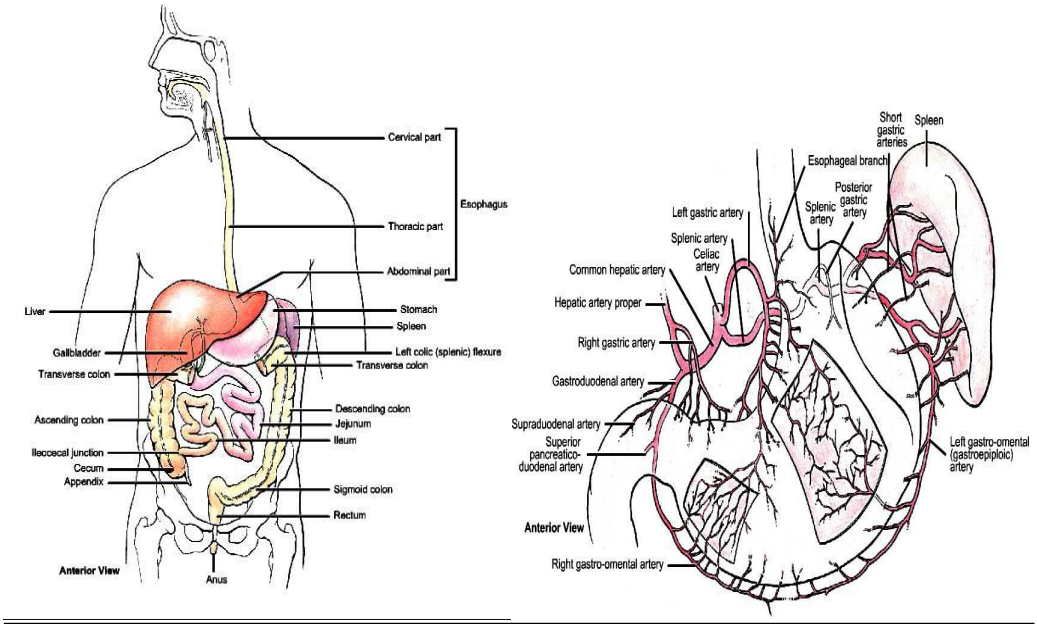


Figure 1 : Gastro intestinal tract

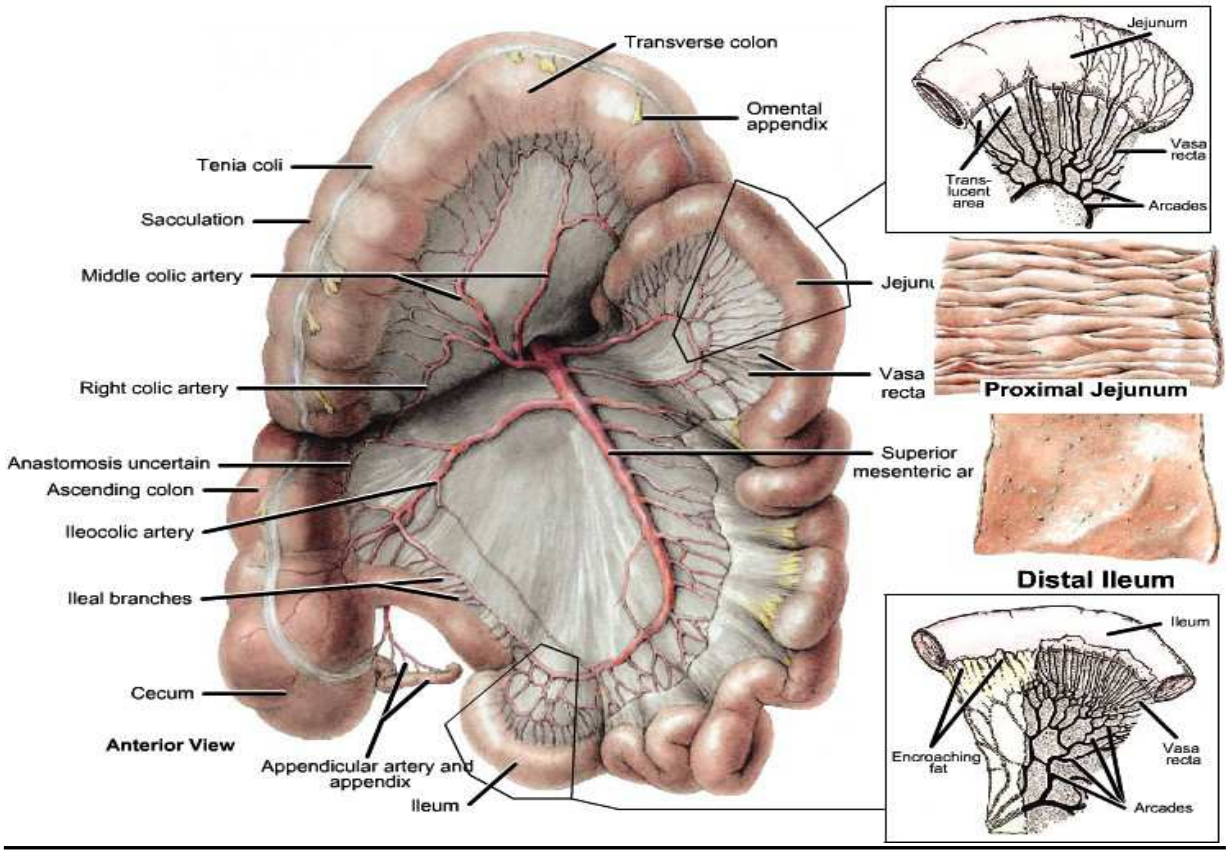


Figure2: Blood supply of small intestine, Mucosal pattern

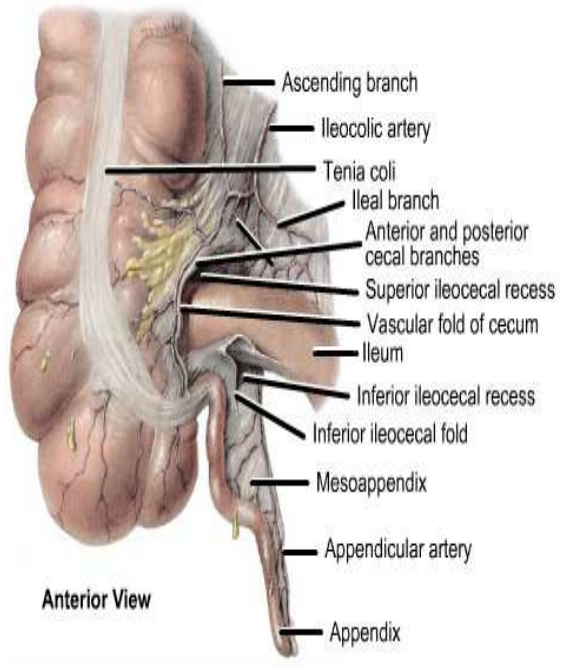


FIGURE: ILEOCAECAL JUNCTION

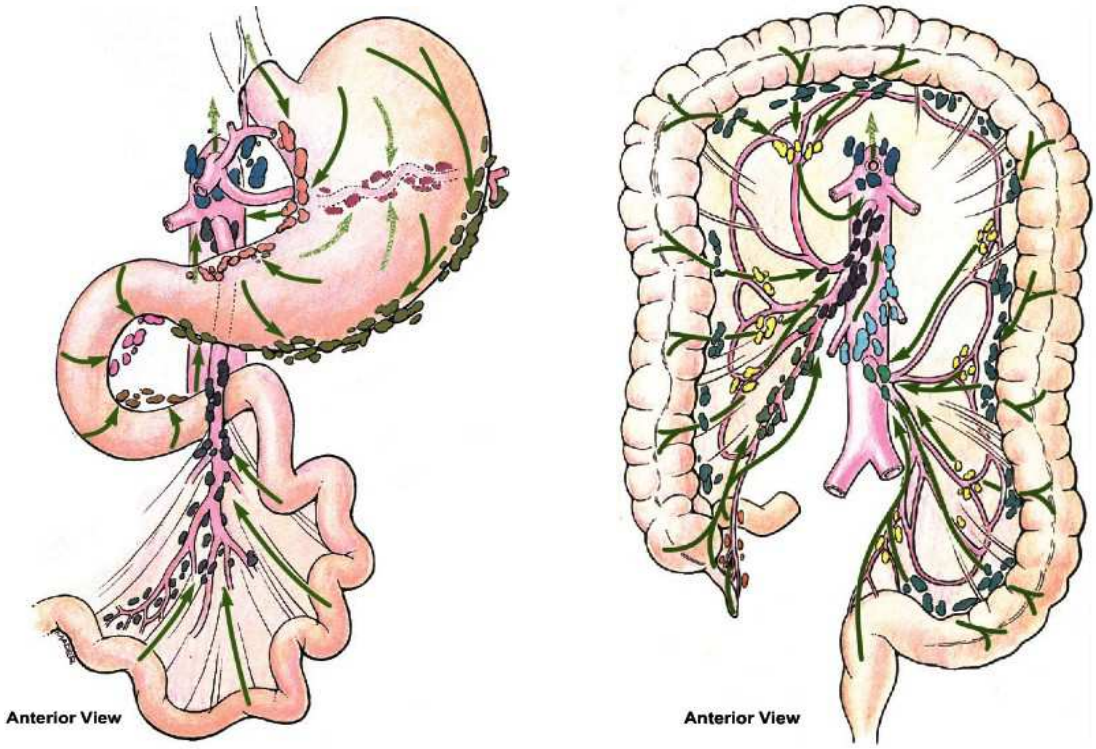


FIGURE: Lymphatic drainage of Stomach & SI

DEVELOPMENTAL ANATOMY

As a result of cephalo caudal and lateral folding of the embryo, the ectoderm lined cavity is divided into intraembryonic portion, the primitive gut, and two extra embryonic portion, the yolk sac and allantois.

In cephalic and caudal parts of embryo, the primitive gut forms a blind ending tube the foregut and hindgut respectively. The middle part forms midgut. The development of gastro intestinal tract is from the following parts of the primitive gut.

Foregut:

It extends from the buccopharyngcal membrane and extending as for caudally as the liver out growth pharyngeal arches differentiates around its cephalic part. Its remaining portion gives rise to oesophagus, stomach, proximal half of the duodenum liver, gall bladder and most of pancreas.

Artery of foregut is coeliac axis.

Midgut:

The midgut grows rapidly that the intra-embroyonic coelum is too small to accommodate it, so that part of loop of extruded into the extraembryonic coelom.

It gives rise to intestines from the middle of duodenum to the junction of right 2/3 and left 1/3 of transverse colon.

The Artery of midgut is superior mesenteric artery.

Hind gut:

It gives rise to the distal third of the transverse colon, the descending colon, the sigmoid colon, the rectum and upper part of anal canal. The terminal part of the hindgut enters into the cloaca.

Artery of hindgut is inferior mesenteric artery.

Rotation of the gut:

Rotation takes place in three stages.

First stage – 5-10 weeks of intra uterine life, the growth of the right lobe of liver exerts pressure on the base of the pre-arterial segment of the midgut loop, so that this segment is pushed down and to the right. This movement forces the post arterial segment upwards and to the left. The first stage of rotation is complete, when midgut loop has rotated through 90 degrees in an anti clockwise direction

2^{nd} stage:

This occurs at 10^{th} to 11^{th} week. At the beginning of the tenth week the midgut loop returns to the abdominal cavity from the umblical cord. The gut being too bulky to be returned enmasse, the pre-arterial portion returns first commencing with its proximal portion. The returning small gut enters the abdomen to the right of superior mesenteric artery but the space here being too limited the coils first reduced are pushed to the left behind the artery by those following on. By their passage to the left they displace the dorsal mesentery of the hindgut before them, so that the descending colon comes to occupy the left flank. The caecum still lies in the umbilical cord on a plane anterior to the small intestine and its artery. The caecum and right half of colon now reduce, passing upwards and to the right of the colon crossing the pedicle of small gut at the point of origin of superior mesenteric artery from aorta and caecum comes to lie under the liver. The subsequent growth elongation of the colon pushes the caecum into the right loin. This completes second stage of rotation.

Third stage:

This occurs between eleventh week and shortly after birth. During this stage

1. Caecum descends further, reaching the right iliac fossa.
2. Certain part of gut becomes fixed to the posterior abdominal wall.

While this process occurs, the midgut loop rotates 270 counter clock wise.

THE DUODENUM

It is shortest, widest and most fixed part of the small intestine[32]. It is 20-25 cm long. It has no mesentery and is partially covered by peritoneum. It is curved around the head of pancreas. Duodenum lies opposite to vertebrae L_1, L_2, and L_3. It is divided into four parts.

I. First part about 5cm long. It begins at pylorus ends at second part of the superior duodenal flexure.

a. Peritoneal relations.

Proximal portion is attached to lesser omentum above and greater omentum below. Distal portion is retroperitoneal

b. Visceral relations.

Anteriorly – Quadrate lobe of liver.

c. Posteriorly – Portal vein, gastroduodenal artery and bile duct.

d. Superiorly – Epiploic foramen

e. Inferiorly – Head of the pancreas.

II. Second part – 7.5 cms long. Begins at superior duodenal flexure, passes downwards to reach lower border of third lumbar vertebrae

a. Peritoneal relations: It is retroperitoneal and fixed, its anterior surface is covered with peritoneum

b. Visceral relations:

Anteriorly – Right lobe of liver, gall bladder and transverse colon Posteriorly – Anterior surface of right kidney near medial border, inferior edge of inferior vena cava and right psoas major muscle.

Medially – Head of pancreas and bile duct. Laterally – right colic flexure.

Interior of 2nd part has major duodenal papilla. It receives the opening of the CBD and pancreatic duct through ampulla of vater. It is situated 8-10cms to the pylorus.

III. Third part – is horizontal, 10cms long.

a. Peritoneal relations–It is retroperitoneal and fixed. Anterior surface is covered by peritoneum except in median plane where it is crossed by superior mesenteric vessels and by the root of mesentery.

b. Visceral relations

Anteriorly – Sup. Mesenteric vessels and root of mesentery

Posteriorly – Right ureter, right psoas, IVC, abdominal aorta and origin of IMA.

Superiorly – Head of pancreas Inferiorly – Coils of Jejunum

IV. Fourth part – about 2.5cms long runs upwards on or immediately left of aorta upto upper border of L2 vertebrae, where it continues as jejunum

a. Peritoneal relations Mostly retroperitoneal

b. Visceral relations

Anteriorly -Transverse colon, transverse mesocolon

Posteriorly – Left sympathetic chain, IMV, left psoas, left renal vessels, left testicular vessels.

Superiorly – body of pancreas.

ARTERIAL SUPPLY

1. Superior pancreatico duodenal artery
2. Inferior panceratico duodenal artery

First part of duodenum receives additional supply from

a. Right gastric artery

b. Supraduodenal artery (of wilkie)

c. Retroduodenal branches of gastroduodenal artery

d. Some branches from right gastroepiploic artery.

VENOUS DRAINAGE

Veins drain in splenic, SMV and portal veins

NERVE SUPPLY

rom celiac plexuses

JEJUNUM AND ILEUM

Jejunum and Ileum is attached to the posterior abdominal wall by mesentery, the proximal of two fifth being jejunum, distal three fourth being the Ileum. The jejunum lies largely in the umbilical region. The jejunum is wider and thicker walled than ileum.

The ileum is thinner than jejunum. It is mainly in hypogastric and pelvic region.

There is no morphological line of distinction between the jejunum and ileum.

Following are some differentiating features.

1. The wall of jejunum is thicker and wider, whereas ileum is thinner and shorter.
2. The fat extending from root of the mesentery does not reach the jejunal border.

Whereas the fat of ileum is thicker and extend fully to the intestinal attachment.

3. Payer's patches are numerous in ileum whereas they are absent in jejunum.
4. Villi are larger in jejunum
5. There is difference in vascular pattern between jejunum and Ileum.

Jejunum – one or two arterial arcades in the mesentery with parallel vessels 3.7cm long going to gut.

Ileum – Two or three arterial arcades as in the mesentery with parallel vessel 1.2cms long going to gut.

Histology

Small intestine has got 4 layers

1. Mucosal layer
2. Submucosal layer
3. Muscular layer
4. Serous layer

1. The mucosal layer – The lining epithelium is columnar all over gut except in esophagus and lower part of anal canal. Mucous membrane is thick and highly vascular in the proximal part thin and less vascular in distal part. There are numerous mucosal folds and at numerous places epithelium dips into lamina propria forming “crypts”. In the small bowel the mucosa has finger like projections called the “villi” and they are scattered in mucosa. Between the bases of villi are the opening of crypts of lieberkuhn.

Three types of cells seen in the epithelium. They are

a. Abortive cells
b. Goblet cells

c. Argentaffin or basal glandular cells.

Lamina propria fills the spaces between the glands of liberkuhn and forms the cover of intestinal villi. The Lamina propria contains many payer's patches.

Payer's patches.

These are aggregates of lymph nodules found mainly in terminal ileum. They are oval in shape and situated opposite the mesenteric attachment. The normal number varies between 30 to 40 and length is 1.2 to 2mms. As the age advances payers patches undergo involution. In typhoid fever, ulceration of these patches may occur. The ulcers are oval in shape and are situated in long axis of bowel.

2. Submucosa:

Mucous membrane rests on layer of loose areolar tissue called submucosa and nerve fibers. It has the highest amount of collagen.

3. Muscular coat:

Composed of plain muscles arranged in two layers. Inner layer is thicker and fibers run circularly. The fibers of outer layer run longitudinally.

4.Serosa:

It is formed from visceral peritoneum invests the gut completely except along the line of mesenteric attachment.

ARTERIAL SUPPLY

Receives blood supply from jejunal and ileal branches of superior mesenteric artery except terminal portion of ileum, which receives from ileocolic artery or from branches of the inferior mesenteric artery.

VENOUS DRAINAGE

Tributaries from ileum and Jejunum drains into superior mesenteric vein.

NERVE SUPPLY

It is from sympathetic T_9-T_{11} as well as parasympathetic (vagus) both pass through the celiac and superior mesenteric plexus.

Sympathetic nerves are motor to the sphincters and muscularis mucosa.

Parasympathetic nerves stimulate peristalsis but inhibit the sphincters.

LYMPHATIC DRAINAGE

Lymphatics run encircling the intestine as the four lacteals pass between the layers of the mesentery. Before reaching the superior mesenteric lymph nodes, the lymph passes through the proximal, intermediate and distal nodes in the mesentery.

PHYSIOLOGY

The physiology of the gastrointestinal system consists of storage, digestion, absorption and propagation of food. Some of these mechanism depend upon the intrinsic

properties of the intestinal smooth muscle others involve the operation of visceral reflexes or the actions of gastro intestinal hormones[34].

SMALL ITESTINE

Here intestinal contents are mixed with the secretion of mucosal cells, pancreatic juice and bile. Digestion is completed and products of digestion are absorbed along with most of the vitamins and fluid.

The contractions of small bowel are coordinated by small bowel wave. The frequency of slow waves decreases from about 12/min in the jejunum to about 9/min in the ileum.

The movements of small intestine mix and churn the intestinal contents (chyme) and propel it towards the large intestine. There are two types of movements, segmentation contractions and peristaltic waves. Both occur in the absence of extrinsic innervation but require an intact myentric nerve plexuses.

REGULATION OF INTESTINAL SECRETION

Brunner's glands in the duodenum secrete thick alkaline mucus that probably helps to protect the duodenal mucosa from the gastric acid. There is also an appreciable secretion of HCO_3 and that is independend stoma. Jejunal perforation can occur in upper part of the jejunum in Zollingent of Brunner's glands secretion but probably has no effect on that of the intestinal glands.

PHYSIOLOGY OF INTESTINAL TRACT:

The major function is absorption which depends upon the complete integration of structural, physiologic and chemical factors. The regulators of gastric biliary, pancreatic and intestinal secretion and motor function provides the appropriate luminal function for complete digestion and presentation of the products of digestion to the intestinal epithelium for absorption and secretion.

The intestinal secretion done by the epithelial cells in the crypts of Lieberkuhn and at the rate of about 1800 ml/day and pH ranging from 7.4--8.0 and contains peptidases, sucrase, maltase, Isomaltase and lactase and lipase.

Absorption is by active transport and diffusion. In digestion and absorption of fluids, electrolytes, iron, folate, carbohydrates, fat and protein is mainly by the duodenum and jejunum. The absorption of bile acids and vitamin B12 occurs in terminal ileum.

The ileum will be able to perform all the functions of jejunum after excision of jejunum. But more length of ileal resection will lead to malabsorption of fat and to diminished bile acid and Vit B12. The other functions of small intestine are that of secreting HDL and LDL which are related to chylomicrons . The GI tract is the largest producer of 1ipoprotiens.

There are endocrine hormones secreted by the small intestine

1) Secretin

2) Cholecystokinin

3) 3) Enteroglucagon

4) Vasoactive intestinal polypeptide

5) Gastric inhibitory polypeptide

6) Motilin

7) Somatostatin

8) Bombesin.

Intestinal motility is mainly by muscular, neural and hormonal factors. There are segmenting contraction makes mixing contraction. Peristalsis on the other hand consists of intestinal contraction . It is slower in distal than in proximal bowel. It has a propulsive function. Under abnormal condition peristaltic rushes can occur, which start from the proximal portion of small bowel or duodenum and rapidly traverse the entire length of the small intestine, followed sometimes by a quiescent period of motor activity.

The intestine also has an immunologic function. It is a source of IgA, which arises from plasma cells in the lamina propria and protein synthesized by the epithelial cells, is secreted into the lumen

Small Intestines Physiology

Digestion

chyme remains in SI for 3-4 hours

Mechanical Digestion

mixes chyme with digestive enzymes

undergoes peristalsis

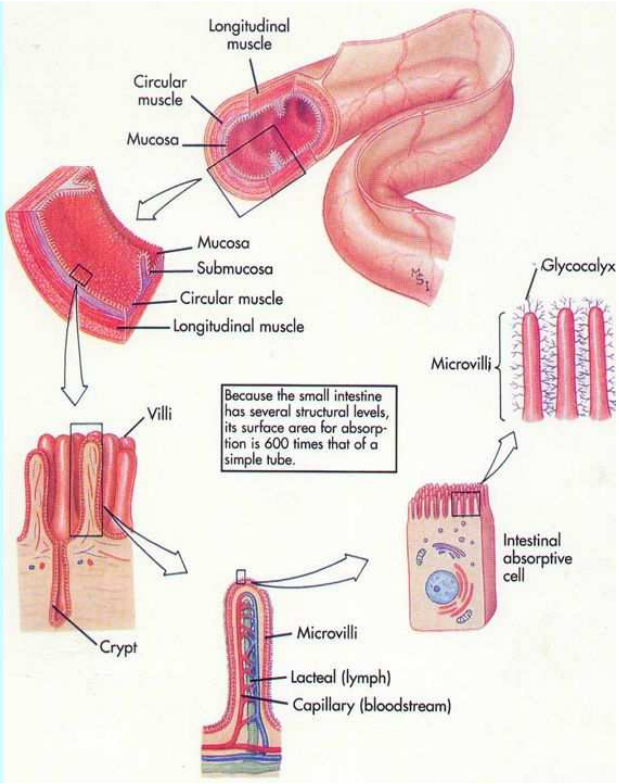
Chemical Digestion

pancreatic juice assists in digestion

Absorption

food products pass through mucosa absorptive cells

passage of these molecules from the GI tract to the blood or lymph is called absorption



Etiology

Small perforation is a serious complication . In developed countries

perforations are reported to be mostly because of foreign bodies, radiotherapy,

crohns disease, drugs, malignancies and congenital malformations. Typhoid fever and TB are rare in developed countries, perforations also rare in these countries.

So the reported case are less in numbers. On the other hand in the underdeveloped countries small bowel perforation is commonly encountered surgical practice. Although TB is an important cause, the most important one is the endemic prevalence of enteric fever in under developed countries.

TYPHOID ENTERITIS

Typhoid fever also known as enteric fever.caused by salmonella enterica. Also seen with subspecies e*nterica* serovar *typhi* and serovars *paratyphi* A, B, and C.

The name *S typhi* is derived from the ancient Greek *typhos*, an ethereal smoke or cloud that was believed to cause disease and madness.

The modes of transmission of enteric fever:

- Oral transmission via food or food handled by asymptomatic individual—a carrier —who sheds the bacteria.
- Hand-to-mouth transmission due to poor hand hygiene
- Oral transmission via sewage-contaminated water

Incidence :

The bowel perforation in enteric fever varies from 0.5% to

78.6% . Studies shows there is alteration in incidence in various countries

Sex and Age

Male preponderance in enteric perforation is common. It is seen in younger age groups. It has been reported in middle age groups. Perforation predominantly occurs in the 2nd and 3rd decades of life.

Seasonal Variation

Studies have shown that more incidence of disease noted at dry seasons.

Pathology

Enteric fever is caused by a Gram-ve bacillus Salmonella typhi. The organism seeps through the **Peyer's** patches. Growth and development occurs in the reticuloendothelial system. Commencement of blood spread noted concurrent with the clinical feautre. During the 2nd week bacteria reach the gut and colonise in **Peyer's** patches. Ulceration and mesenteric adenitis seen. . Perforation is commonly seen in the second week following onset of illness.

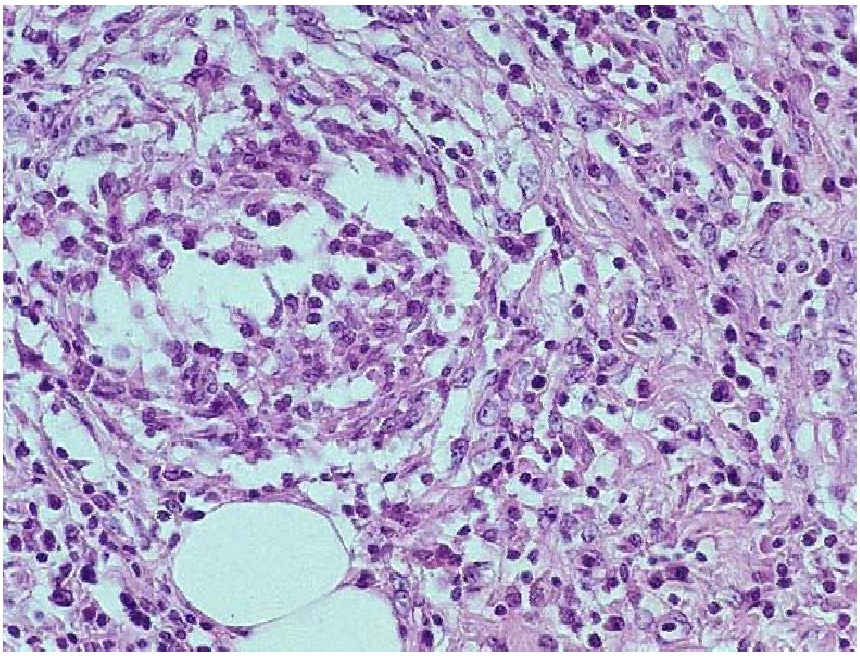
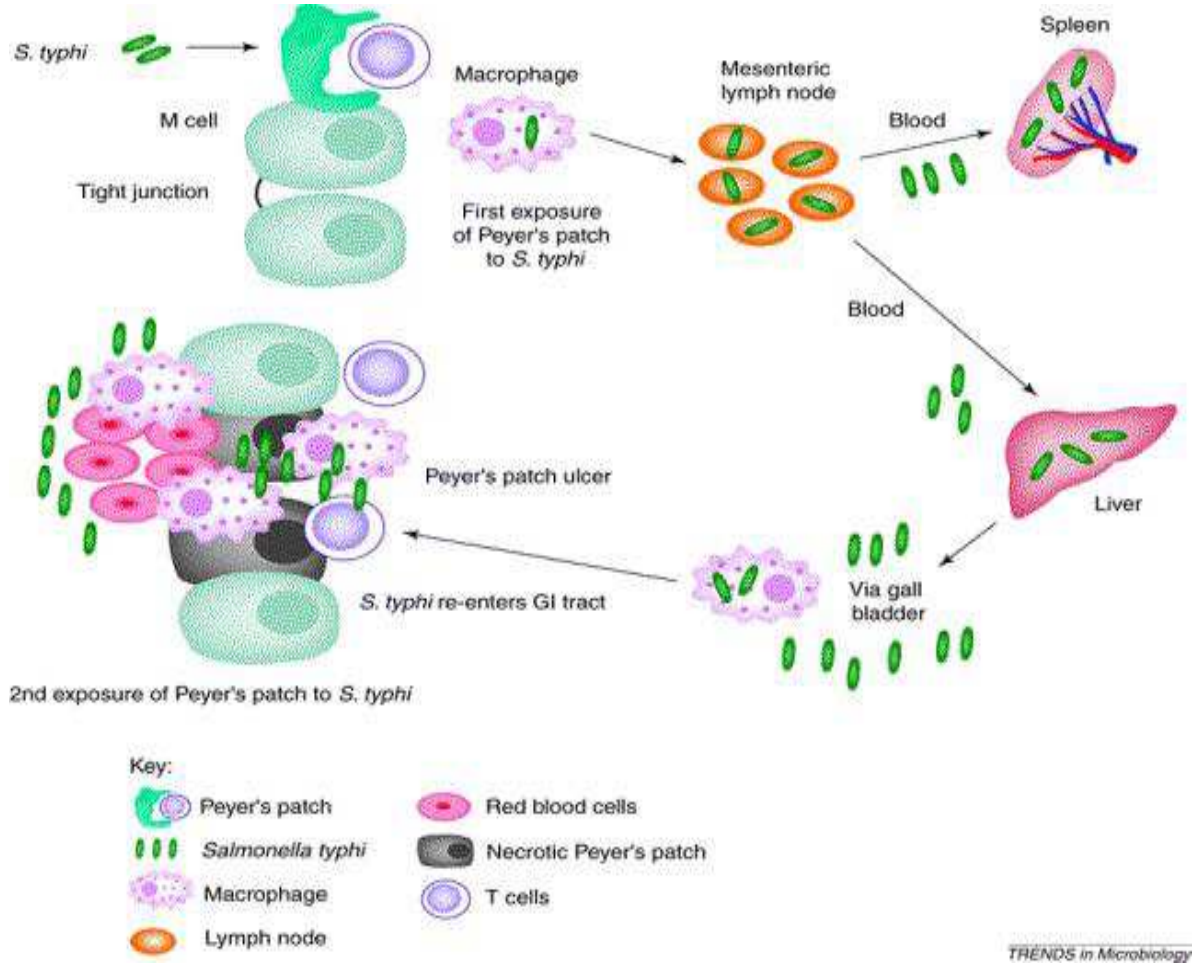


FIGURE 5: HISTOPATHOLOGY IN TYPHOID ILEAL PERFORATION



Macroscopy

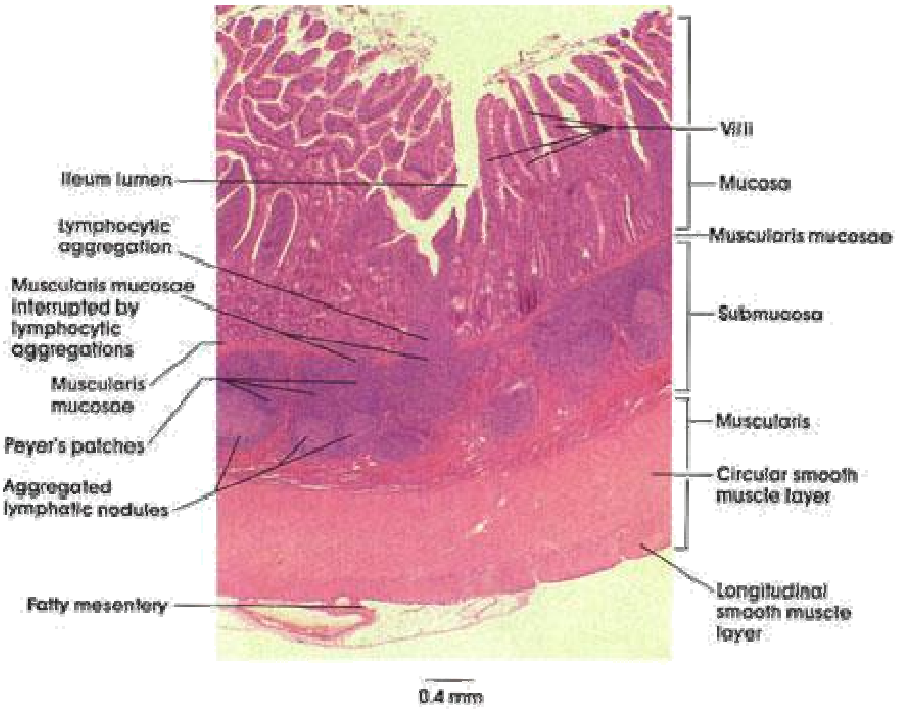
Peyer's patchesbecomes prominent. Mesenteric nodes are enlarged. The terminal ileum are most commonly affected. Ulceration occurs in the long axis of the bowel.

Multiple perforations are usually seen

Most of the perforations occur within 30cm of ileocaecal junction

Microscopy

There is marked proliferation of reticuloendothelial cells of the lymphoid follicles. There are mononuclear phagocytes seen. Erythrophagocytosis is noted . The bacteria are rarely visualized.



Aetiopathogenesis

Typhoid ulcer caused by Salmonella typhi and paratyphi occurs in the terminal ileum. The ulcers are arranged parallel (longitudinal) in relation to the bowel wall. Endarteritis and concomitant necrosis can vary in size and cause spillage of bowel contents into the peritoneal cavity. The more insidious the perforation, greater is the incidence of developing pericolic abscess due to the “walling effect” by adhesions

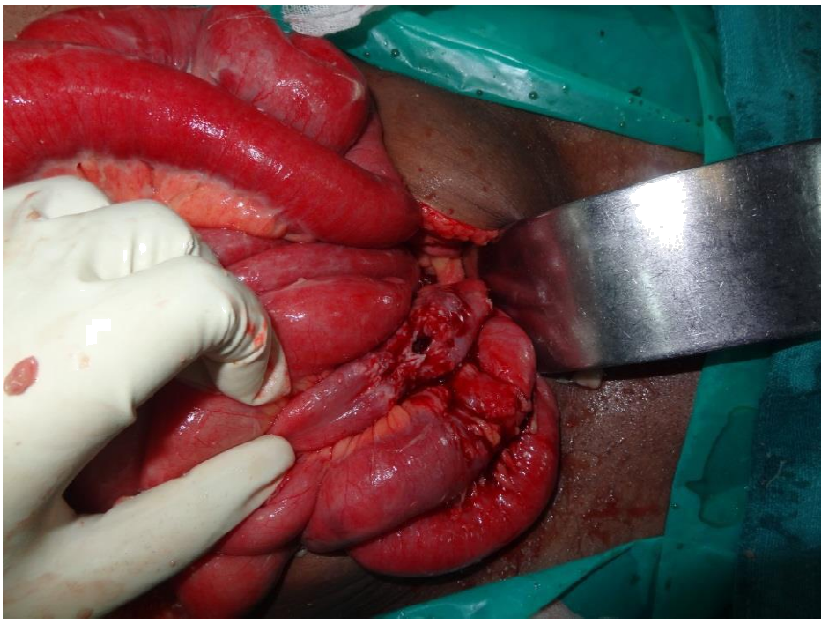
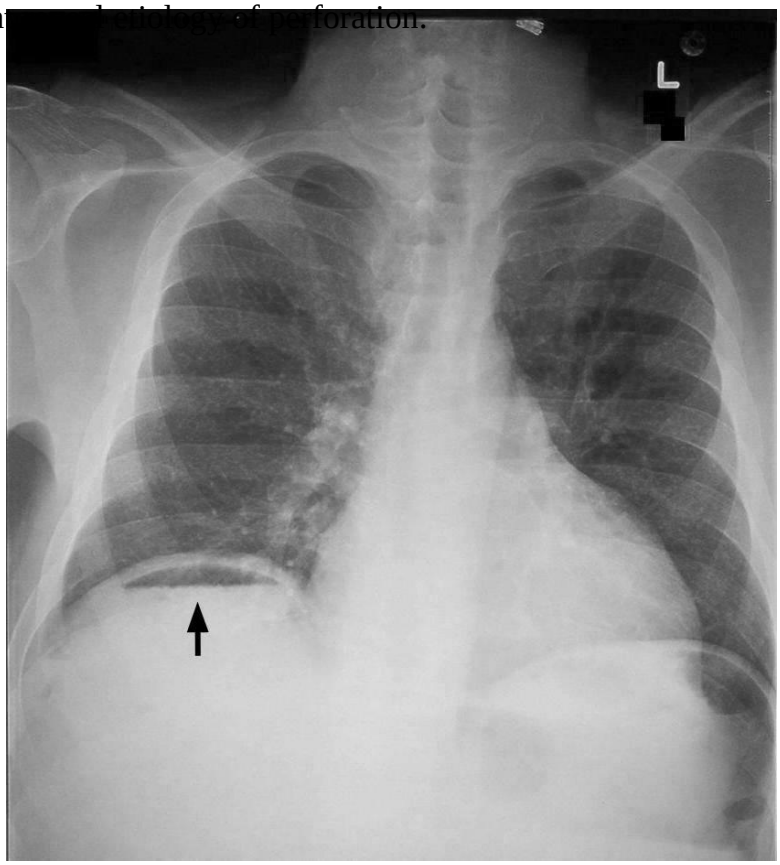


FIGURE: ILEAL PERFORATION

Diagnosis

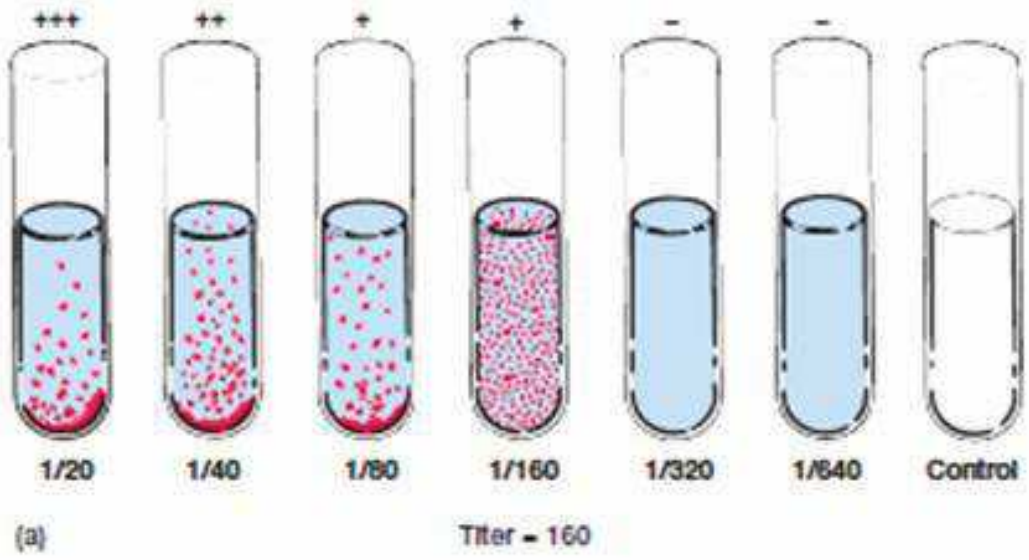
Clinical suspicion can be made out in enteric fever condition . Air may be present under the diaphragm. Pneumoperitoneum are seen in majority of cases. Abdominal paracentesis may reveal fluid for analysis. . Peritoneal lavage are also done to detect bile or pus

The diagnosis of typhoid fever can be made by Widal test, blood culture. New diagnostic techniques have been introduced for rapid diagnosis of enteric fever. Histopath analysis of the biopsy migh[illegible] etiology of perforation.



Serology

Widal test done to detect antibodies against the flagellar and capsular antigens of the salmonella species. Diagnosis accurately made from seventh to tenth day. Salmonella typhi analysis can be made from blood, bone marrow aspiration stool and urine.



Newer Methods

Indirect heamagglutination, indirect fluorescent Vi Antibody and ELISA are the current newer methods. The use of monoclonal antibodies and DNA probes for detection of Salmonella typhi can also be done. Newer techniques give rapid results

TUBERCULOUS PERFORATION

One would not be wrong in considering tuberculosis as a differential diagnosis for most abdominal pathologies in the East and perforation is one amongst them.

Primary intestinal tuberculosis (without pulmonary involvement) is the commonest forms of extra pulmonary tuberculosis. It is caused by ingestion of contaminated milk that leads to a primary infection of the intestine in the absence of pulmonary disease. Intestinal TB commonly affects the ileocaecal region because of the following reasons:

1) ileum is an area of stasis;

2) it has abundant lymphoid tissue

3) it has a high absorptive capacity.

Infection occurs in the Peyer's patches, mucosal edema and slough out noted , leading to the formation of ulcers that lie transversely to the long axis of the ileum. The disease may spread and heal by fibrosis

Aetiopathogensis : [28,29]

It is better to consider this in relation to the types of ileocaecal tuberculosis (most common area involved in intestinal tuberculosis).

a. Ulcerative – The ulcers are perpendicular to the longitudinal axis of the bowel in the direction of the lymphatics. It arises in most instances secondary to pulmonary Koch's. The omental serosa is studded with tubercles. The associated fibrosis reduces the chance of perforation to less than 1% (Bhattacharya)[30].

b. Hyperplastic – Due to previous immunity to tuberculosis with ingestion of Mycobacterium bovis strain in milk, there is thickening of the wall and lymphadenitis.

c. Ulcero – hyperplastic – Clinical features are anorexia, asthenia, dyspepsia, blood and mucous in stool, intestinal obstruction, symptoms of blind loop syndrome and signs of peritonitis with doughy abdomen, tenderness in the right iliac fossa. There is a 12-18% association with pulmonary Kochs.

FIGURE : TUBERCULAR PERFORATION SHOWING STRICTURE AND PERFORATION PROXIMAL TO STRICTURE

ASCARIASIS ROUND WORM INFESTATION

Ascariasis lumbricoides is extremely common in warm climate and in the most common parasite found in human beings. Surgical complications like obstruction and perforation may arise.

Perforation due to round worm is encountered uncommonly, usually the round worm are not the primary cause of perforation. The perforation occurs either due to pressure necrosis of wall by impacted worms or necrosis due to some toxin liberated by parasite.

FIGURE : ROUND WORMS REMOVED BY ENTEROTOMY

PERORATION OF MALIGNANT TUMOR OF SMALL INTESTINE

Compare with large intestine, small intestine is rarely the seat of a neoplasm, and these become progressively less common from duodenum to terminal ileum. These comprise 2% of all gastro intestinal tumours. Adenocarcinoma is more frequent followed by sarcomas. Adenocarcinoma occurs with equal frequency in duodenum, jejunum & ileum, where as sarcoma occur predominantly in ileum.

Perforations of malignant tumors are very rare. Perforation may occur with or without obstruction. Prognosis is bad in these cases because diffuse peritonitis is badly tolerated by a patient with malignant disease small bowel perforation is also a rare complication of met static lung cancer[42]. small bowel tumors in order of frequency are adenocarcinoma, carcinoid, lymphoma and sarcoma.

Lymphomas are the commonest small bowel tumors to perforate.Most common near ileum.This may occur in an area of cancerous involvement often secondary to partial or complete distal obstruction

Inflammatory Bowel Disease

Crohn's disease is transmural inflammation of the bowel. The fibrous reaction and adherence to surrounding structure limit the complication of free perforation.it present as free perforation or secondary perforation abscess condition. Free bowel perforation is an indications for emergency surgery . complication is rare,usually don’t need an emergency surgery.treated with medication.

Non-specific Perforation

When the etiology of small bowel perforation is not identified, it is called as a non-specific perforation. It is due to undiagnosed typhoid or other causes such as diet, drugs, viral or parasitic infections. It was earlier thought to undiagnosed typhoid but outcome different when compared to those with typhoid perforation. It has been found that the sub mucosal vascular emboli may be responsible for such perforations.

Diverticulitis

Meckel's diverticulum is located in the distal ileum, found within 60 cm

(2 feet) of the ileocecal valve. The pouch is about 3–

6 cm long and have a lumen diameter more than that of the ileum.. it is seen in antimesentericborder and has its own blood supply.

It is a remnant ofof the yolk sac present during embryonic development. It is a *true diverticulum*, consisting of all 3 layers of

the bowel wall (mucosa, submucosa and muscularis propria.)

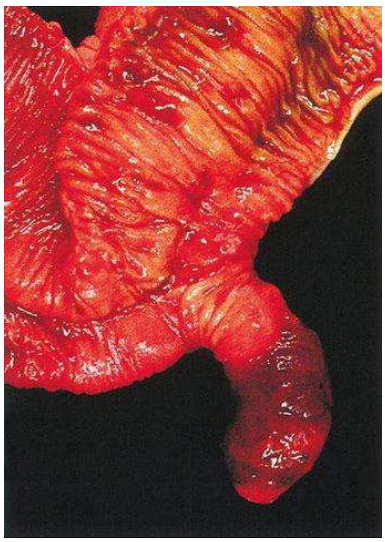
Meckel's diverticulum may contain abnormal tissues, containing other tissue types. Jejunal, duodenal mucosa or Brunner's tissue Heterotopic rests of gastric mucosa and pancreatic tissue are seen .Inflammation of this Meckel's diverticulum mimic appendicitis.

the rule of 2s:

- *2%* (of the population)
- *2* feet (proximal to the ileocecal valve)
- *2* inches (in length)
- *2* types of common ectopic tissue (gastric and pancreatic)
- *2* years is the most common age at clinical presentation
- *2*:1 male:female ratio

Perforation of diverticula is a rare cause of small bowel perforation.

Meckel's diverticulum occurs in 2%population. Gastric mucosa is found in up to 38% of **Meckel's** diverticula .Perforation of diverticulum is rare. The gastric mucosa in a **Meckel's** diverticulum may lead to ulceration, which might perforate.



ISCHEMIC ENTERITIS

Ischemic enteritis is a rare cause of small bowel perforation.

The gross lesion can be described in four stages

1. Segmental bluish discoloration,

2. Circular purple bands

3. Segment becomes longer, pipe-like

4. The segment becomes thin

Perforation usually occurs in the fourth stage.

Miscellaneous

The miscellaneous causes reported are roundworm infestations, polyarteritis nodosa, radiation enteritis, steroid dependency, and AIDS .Steroid theraphy in myeloproliferative disorders, connective tissue disorders and metastatic cancer are at risk. The risk of perforation seen highest during the first three weeks of starting steroid therapy

MATERIALS AND METHODS

MATERIALS AND METHODS

Study Centre

Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. A minimum of 103 patients of small bowel perforations included in the study.

Duration of Study

May 2017 to October 2018

Study Design

Prospective study (Observational)

Inclusion Criteria

The patients admitted to various surgical wards in RGGGH evaluated as SMALL BOWEL PERFORATION

Exclusion criteria

Duodenal perforation (D1D2)

Large bowel perforation

Previous irradiated abdomen

Trauma

Ethics Clearance

Obtained

Methodology

103 consecutive patients who fit the inclusion criteria will be studied and the following data collected

All patients who fulfill the inclusion criteria will be enrolled.

Written informed consents will be obtained.

Opened for laprotomy ,small bowel throughly examined from duodenum to ileum .perforation edge biopsy taken send for HPE

Peritoneal fluid is taken and sent for cytology and bacteriology staining

Stastical Analysis

All the collected data were tabulated on MS Excel sheet .All the above collected data will be analysed and conclusions will be derived through statistical analysis

RESULTS

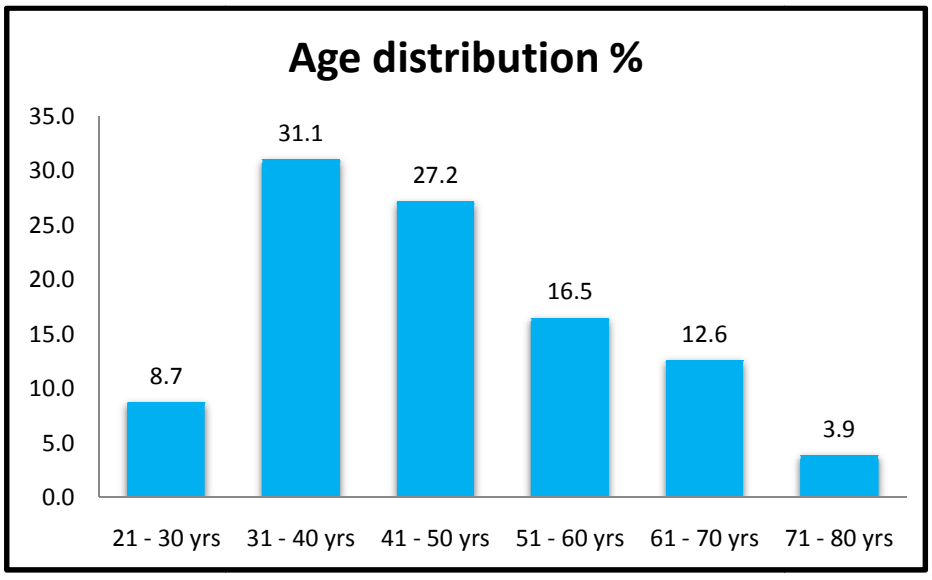
RESULTS

The study was conducted in Institute of general surgery, RGGGH between May 2017-October 2018. The total study population was 103 who met the criteria for inclusion in the study.

AGE

The age of patients ranged from 21 to 80 years. Perforation commonly occurred in the third and fourth decades of life with 58.3% of patients between the ages of 30 and 50.

		Frequency	Percent
Valid	21 - 30 yrs	9	8.7
	31 - 40 yrs	32	31.1
	41 - 50 yrs	28	27.2
	51 - 60 yrs	17	16.5
	61 - 70 yrs	13	12.6
	71 - 80 yrs	4	3.9
	Total	103	100.0

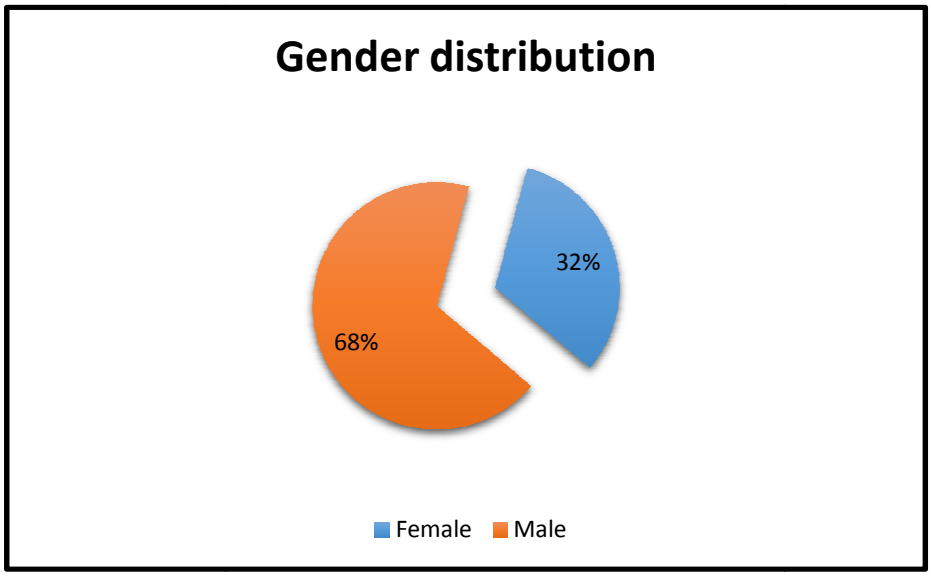


SEX

Incidence of male sex is found to increased with 68% followed by female sex with 32%

SEX

		Frequency	Percent
Valid	Female	33	32.0
	Male	70	68.0
	Total	103	100.0

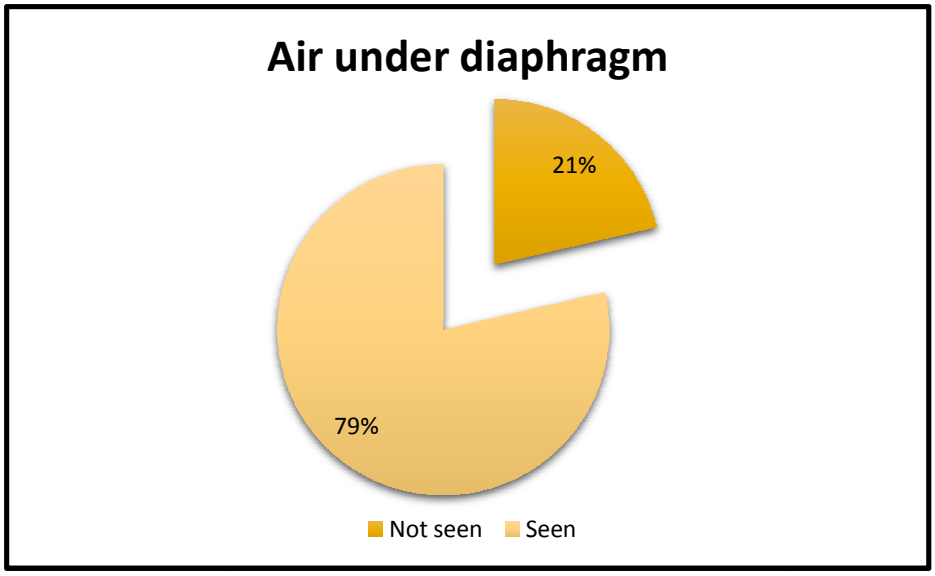


AIR UNDER DIPHRAGM

X-Ray: Pneumoperitoneum in chest and erect abdominal x-ray was seen in 78.6% of patients.

AUD

		Frequency	Percent
Valid	Not seen	22	21.4
	Seen	81	78.6
	Total	103	100.0



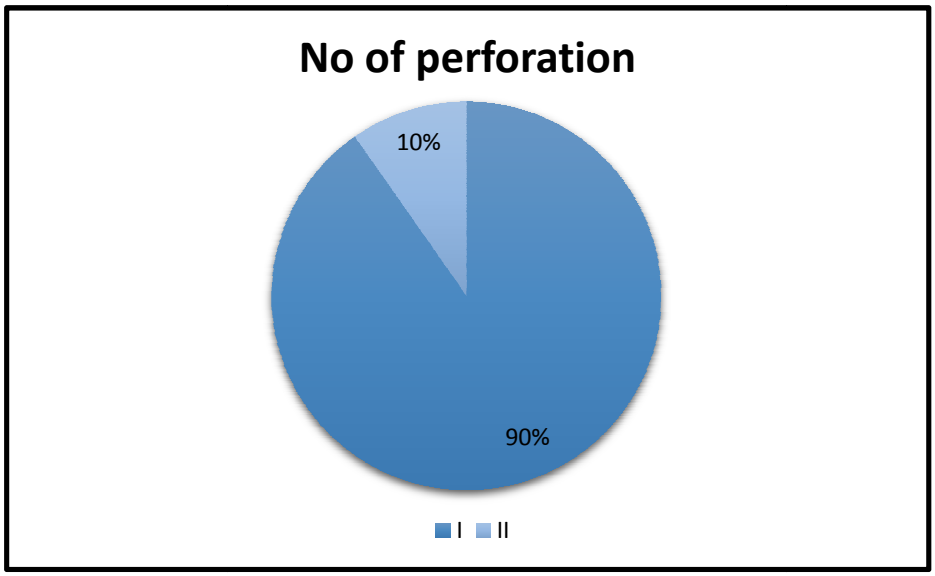
NUMBER OF PERFORATION

Analysis of perforation respective to the number and location.

There were incidence of 93 patients present with single perforation and 10 patients present with multiple perforation which is predominantly typhoid etiology

NO OF PERF

		Frequency	Percent
Valid	I	93	90.3
	II	10	9.7
	Total	103	100.0

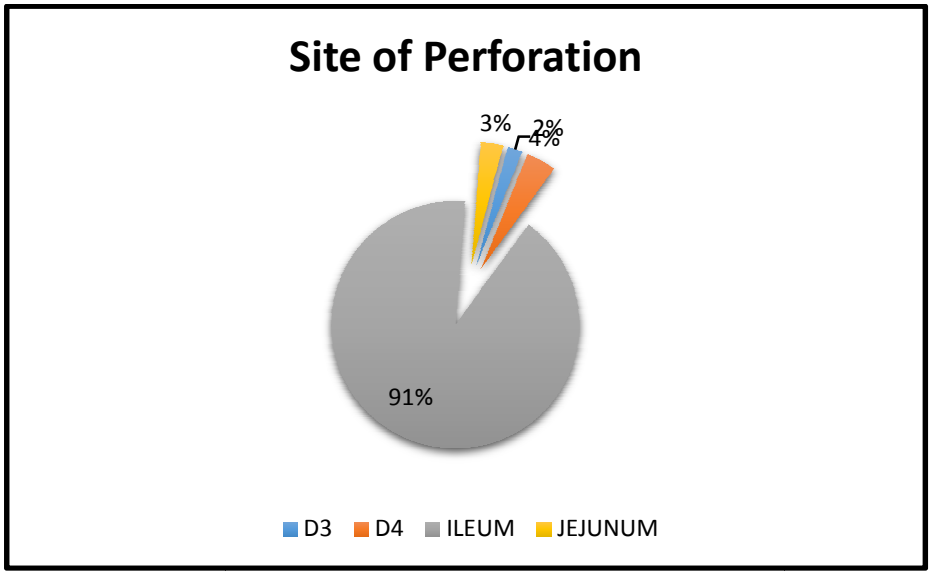


SITE OF PERFORATION

most common site for perforation is ileum with 94 patients follwed by 4th part of duodenum, jejunum and 2nd part of duodenum.

SITE OF PERF

		Frequency	Percent
Valid	D3	2	1.9
	D4	4	3.9
	ILEUM	94	91.3
	JEJUNUM	3	2.9
	Total	103	100.0

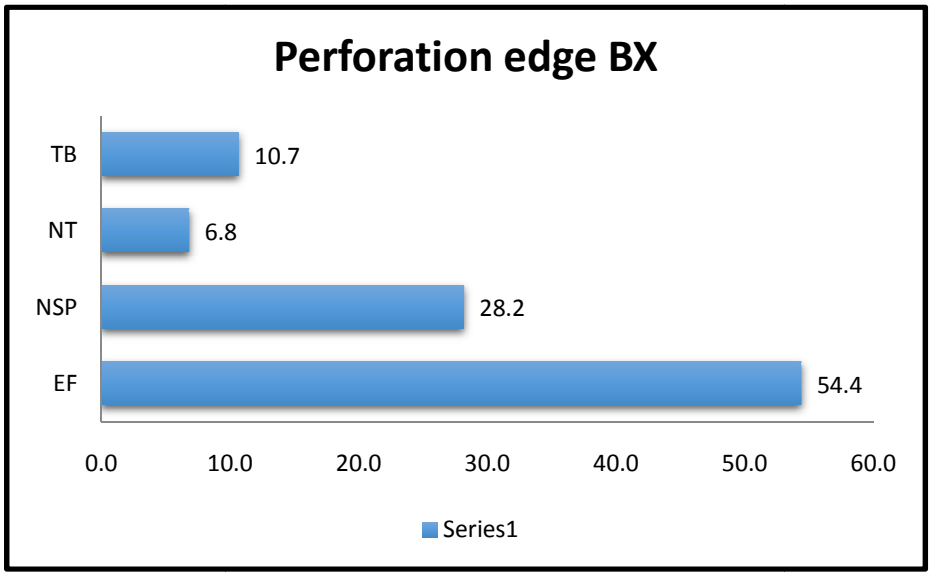


PERFORATION EDGE BIOPSY

Pathological examination of either resected specimens or scrapings from the edge of the ulcer was done in 96 patients. A report suggestive of typhoid was seen in 56 cases. A diagnosis of tuberculosis was made in 11 cases and the rest showed features of non-specific inflammation with no conclusive diagnosis.

PERF EDGE BX

		Frequency	Percent
Valid	EF	56	54.4
	NSP	29	28.2
	NT	7	6.8
	TB	11	10.7
	Total	103	100.0

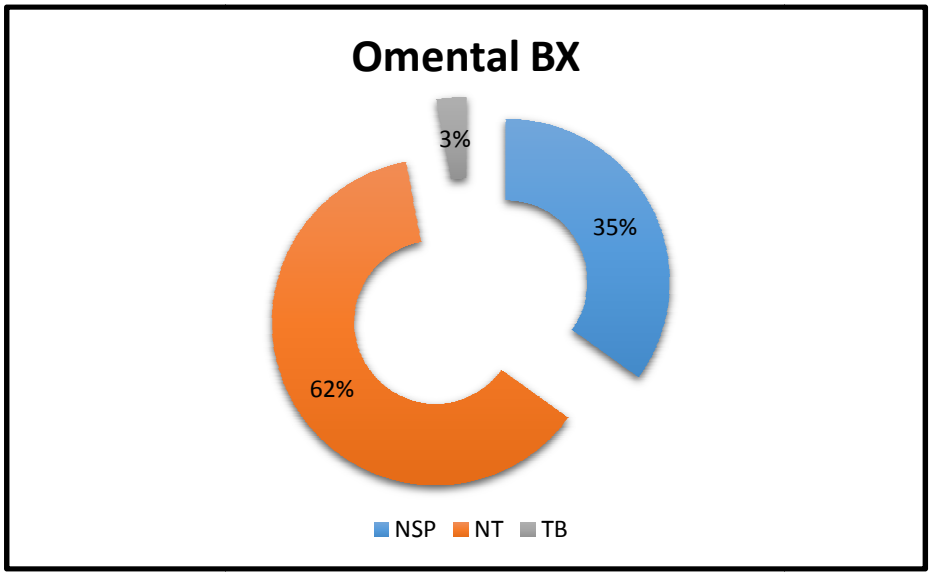


OMENTAL BIOPSY

Omental biospsy was taken for 39 patients. Reports suggestive of tuberculosis in 3 patients. Rest showed non specific pathology.

OMENTAL BX

		Frequency	Percent
Valid	NSP	36	35.0
	NT	64	62.1
	TB	3	2.9
	Total	103	100.0

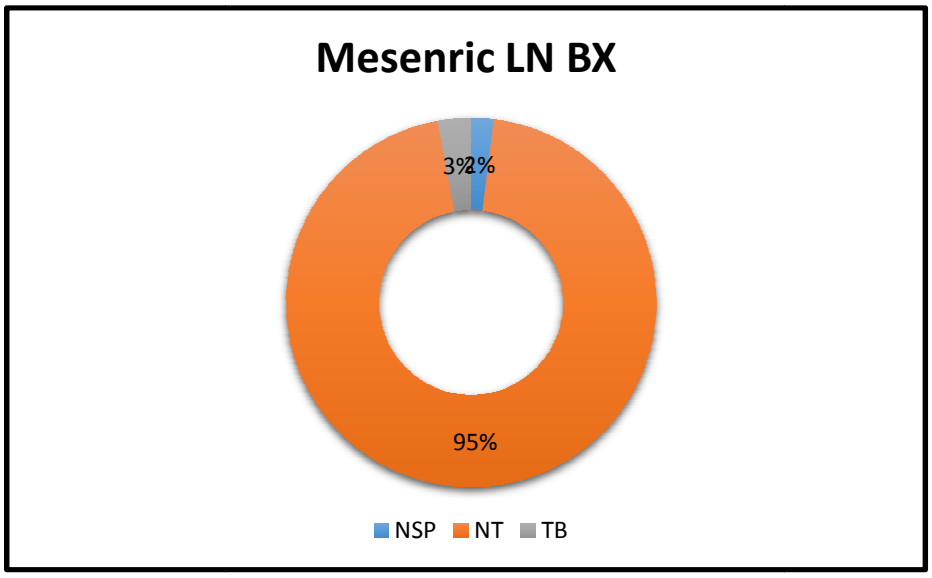


MESENTERIC LYMPHNODE BIOPSY

Mesenteric lymph node analysis was done in 5 patients result shows 3 patients positive for tuberculosis and 2 positive for non specific pathology.

MESENTERIC LYMPHNODE BIOPSY

		Frequency	Percent
Valid	NSP	2	1.9
	NT	98	95.1
	TB	3	2.9
	Total	103	100.0

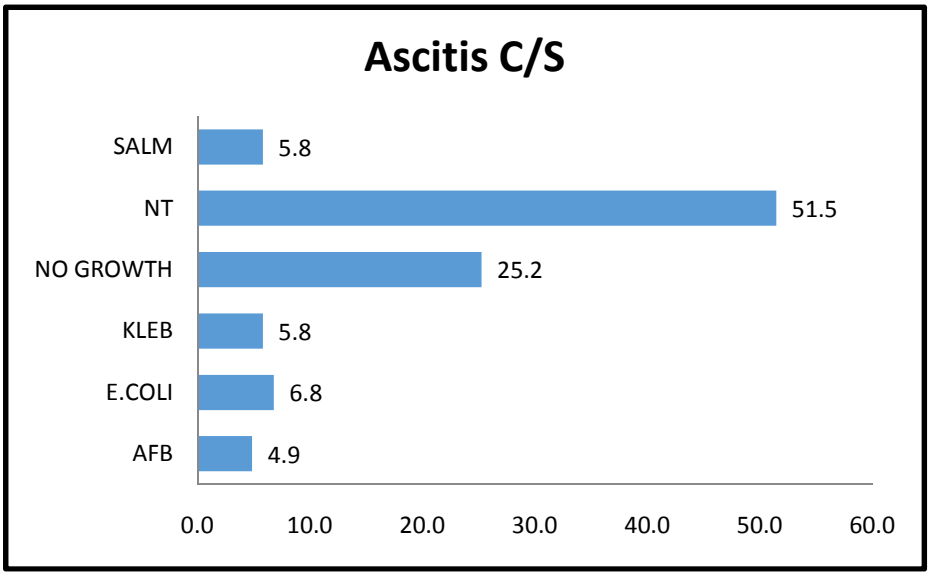


ASCITIC CULTURE/SENSITIVITY

ASCITIC CULTURE/SENSITIVITY was done in 50 patients results Report suggestive of salmonella in 6 pts, E.coli in 7 pts,klebseilla in 6 patients,afb for 5 patients,no growth in 26 patients.

ASCITIC CULTURE/SENSITIVITY

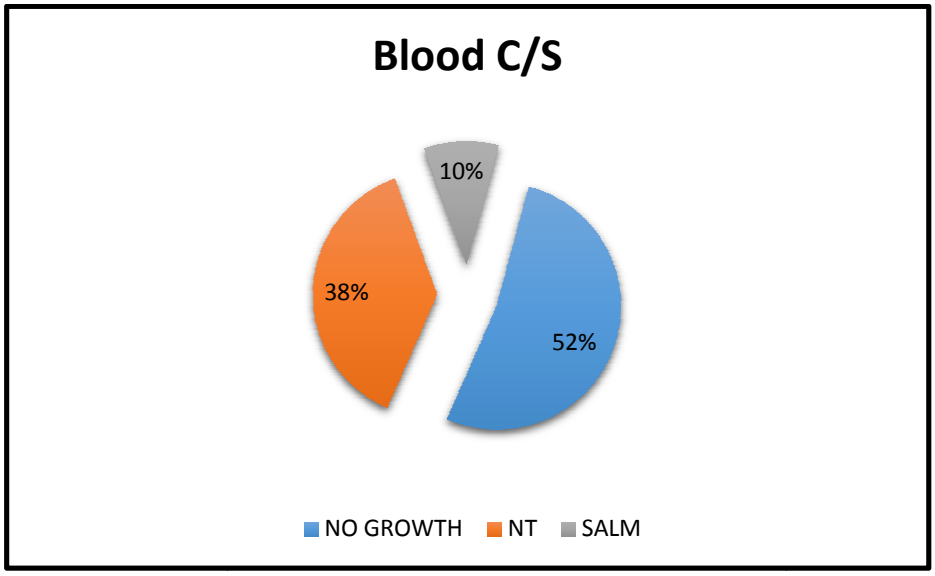
		Frequency	Percent
Valid	AFB	5	4.9
	E.COLI	7	6.8
	KLEB	6	5.8
	NO GROWTH	26	25.2
	NT	53	51.5
	SALM	6	5.8
	Total	103	100.0



BLOOD CULTURE/SENSITIVITY

Blood cultures were done in 64 patients and Salmonella typhi was grown in 10 patients rest shows no growth..

		Frequency	Percent
Valid	NO GROWTH	54	52.4
	NT	39	37.9
	SALM	10	9.7
	Total	103	100.0

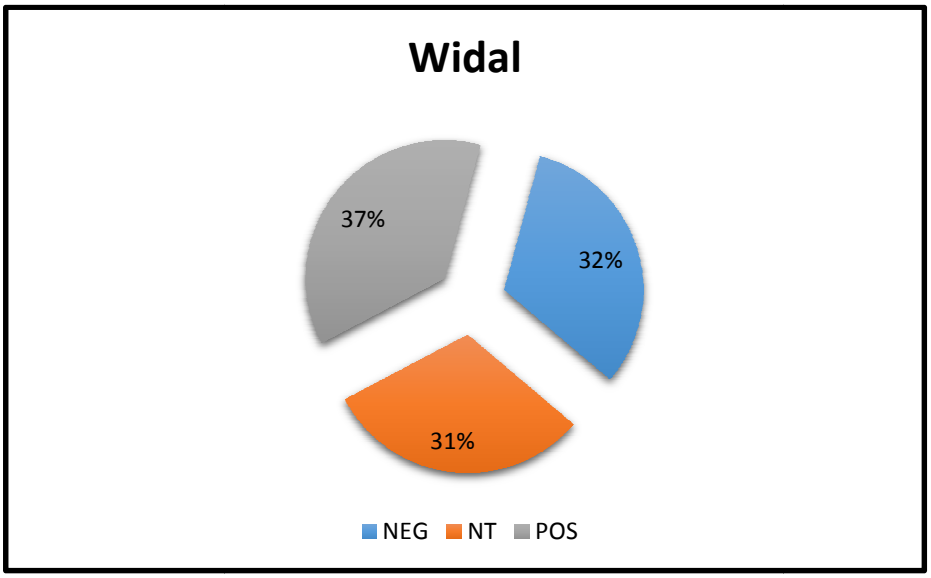


WIDAL TEST

Widal test was positive in 38 patients out of 71 patients of enteric perforation.

WIDAL

		Frequency	Percent
Valid	NEG	33	32.0
	NT	32	31.1
	POS	38	36.9
	Total	103	100.0



DISCUSSION

DISCUSSION

The commonest cause of small bowel perforation in the study was typhoid fever accounting for 54% of cases. Enteric fever was the commonest cause of ileal perforation in tropical countries and the study also shows the same results. Enteric fever accounted for 56.6% of cases of ileal perforation in the series by Karmakar. Mechanical causes and malignancy are the causes of small bowel perforation in the western counteries . Mechanical causes and lymphomas are seen in 40.7% of perforations in the series by Dixon and Orringer . There were less number of typhoid perforation cases in either series from western countries .When the cause of the perforation was not identified it was termed non-specific perforation. Non-specific perforation was the second commonest cause in this study accounting for 28.2% of cases. Widal test, blood culture and histopathology were suggestive of typhoid. Non-specific perforations were the commonest cause of small bowel perforation in the series by Dixon and Bhalerao. TB accounted for 10.7% of cases of small bowel perforations in this study. Talwar et al have found 19% of nontraumatic small bowel perforations were due to intestinal TB. In 90% of the cases, perforation is solitary. The age of patients ranged from 21 to 80 years. Perforation commonly occurred in the third and fourth decades of life with 58.3% of patients between the ages of 30 and 50. Incidence of male sex is found to increased with 68% followed by female sex with 32% Patient usually present with ulcer in 2 to 3rd week of disease.

Typhoid perforations as reported by Eggleston occurred in the second and third decades of life. Keenan reported that 88% of patients perforated in the second week . Lizzaralde reported that 54.2% of patients perforated in the second week .Chest X-ray is a useful investigation to detect hollow viscus perforation. Pneumoperitoneum has been reported in 52% to 82% in studies by Hadley. Pneumoperitoneum in chest and erect abdominal x-ray was seen in 78.6% of patients. Widal was reported positive in 30% of patients with typhoid perforation by Kaul and in 46.1% of patients by Santillana

Blood cultures were done in 64 patients and Salmonella typhi was grown in 10 patients rest shows no growth.. Widal test was positive in 38 patients out of 71 patients of enteric perforation..Tuberculosis was diagnosed definitively by histopathology. Histopathology was suggestive of typhoid in all enteric perforation patients. The presence of erythrophagocytosis virtually confirms the diagnosis of typhoid perforation.. Widal along with HPE is the most useful test for typhoid. It is easily available and is less susceptible to prior therapy when compared to blood culture. This usefulness was confirmed by Jarrett. Pathological examination of either resected specimens or scrapings from the edge of the ulcer was done in 96 patients. A report suggestive of typhoid was seen in 56 cases. A diagnosis of tuberculosis was made in 11 cases and the rest showed features of non-specific inflammation with no conclusive diagnosis.

Omental biospsy was taken for 39 patients. Reports suggestive of tuberculosis in 3 patients. Rest showed non specific pathology. Mesenteric lymph node analysis was done in 5 patients result shows 3 patients positive for tuberculosis and 2 positive for non specific pathology. Ascitic c/s was done in 50 patients results Report suggestive of salmonella in 6 pts, E.coli in 7 pts,klebseilla in 6 patients,afb for 5 patients,no growth in 26 patients.Analysis of perforation respective to the number and location. There were incidence of 93 patients present with single perforation and 10 patients present with multiple perforation which is predominantly typhoid etiology and the most common site for perforation is ileum with 94 patients followed by 4^{th} part of duodenum, jejunum and 2^{nd} part of duodenum.

SUMMARY AND CONCLUSION

SUMMARY

Small bowel Perforation commonly occurred in the third and fourth decades of life with increased incidence with male sex. Air under diaphragm was seen in most of the cases. Blood c/s and widal test was in favour of typhoid perforation. Perforation biopsy report suggestive of typhoid in majority of cases. Omental biopsy shows non specific pathology in most of the cases and ascitic fluid analysis was seen in favour of tuberculosis and the most common site of perforation was found to be ileum.

Conclusion

- Typhoid is the most common cause of small bowel perforation, followed by non-specific perforations and tuberculosis.
- Patients have a male preponderance and usually in the 3rd and 4 th decades of their lives.
- Widal serology is a useful test in the diagnosis of typhoid fever. Histopathology is useful in the diagnosis of tubercular perforations and very useful in the diagnosis of typhoid.

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ANNEXURES

PROFORMA

DATA COLLECTION SHEET

I.Patient particulars:

Name	DOA	Case No.
Age	DOS	I.p.No.
Sex	DOD	Address

Occupation:

II.Diagnosis

III.Chief complaints (with duration)

A. Abdominal pain

B. fever

C.Other complaints

PAST HISTORY:

HISTORY OF PREVIOUS OPERATION -

HISTORY OF PREVIOUS IRRADIATION

DURATION OF DIABETES -

PERSONAL HISTORY:

CLINICAL EXAMINATION:

Pulse :	BP :
RR :	Temp :
Pallor :	Icterus :
CVS :	RS :
P/A :	CNS:

VII.MANAGEMENT:

MODE OF TREATMENT – CONSERVATIVE / SURGICAL PROCEDURE WITH INTRA OPERATIVE FINDING

VIII.COMPLICATIONS:

IX.FOLLOW UP:

BLOOD C/S

WIDAL TEST

ULCER EDGE BIOPSY,

MESENTERIC LYMPH NODES

OMENTAL BIOPSY

PERITONEAL ASPIRATE were subjected to culture to determine the predominant aerobic bacterial isolate and its antibiogram.

INFORMATION SHEET

We are conducting a study on" ANALYSIS OF RISK FACTOR AND ETIOLOGY IN ACUTE ABOMEN WITH SMALL BOWEL PERFORATION"

among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your information is valuble to us.

The purpose of this study is to assess the magnitude of problem and evaluate the cause leading to small bowel perforation in acute abdomen at RGGGH,Chennai.
We are selecting certain cases and if you are found eligible, we may be using your information which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the Participant Signature of the Investigator

Date

Place

PATIENT CONSENT FORM

STUDY TITLE:

"ANALYSIS OF RISK FACTOR AND ETIOLOGY IN ACUTE ABOMEN WITH SMALL BOWEL PERFORATION"

STUDY CENTRE:

Rajiv Gandhi Government General hospital and Madras Medical College.

PARTICIPANT NAME: AGE: SEX: I.P. NO :

I confirm that I have understood the purpose of interventional procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the interventional and interventional procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that the investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study of the SMALL BOWEL PERFORATION IN ACUTE ABDOMEN REDEFINING ITS CAUSE

Date: signature / thumb impression of patient

Place:

Patient's name:

Signature of the Investigator: _____

Name of the investigator:

CERTIFICATE – II

This is to certify that this dissertation work titled

“SMALL BOWEL PERFORATION IN ACUTE ABDOMEN -

REDEFINING ITS CAUSE”

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